بسم الله الرحمن الرحيم

والحمد لله رب العالمين والصلاة والسلام علي أشرف المرسلين سيدنا محمد النبي الأمي الهادي الأمين وعلي آله وصحبه أجمعين

وبعـــــد

أخي الحبيب .... أختي الكريمة :

والله إني أحبكم جميعا في الله ....

وما حملني علي كتابة هذه الكلمات إلا ابتغاء مرضاة الله ثم لعلها تنفع واحدا منكم في امتحانه أو في حياته فيما بعد ...

فمن وجد شيئا يعجبه فليدع لنا بالقبول والاخلاص ومن وجد شيئا لم يعجبه فلينصحنا سرا ولا يفضحنا..

ولا تنسوا الدعاء لاخواننا الكرام د. خالد عيد و د. معاوية عصام وأخواتنا الكريمات د. فتحية الحسيني و د. أمل أيوب و د. سمر عادل و د. مها علام و د. ريهام امام و د. الهام . تقبل الله منهم جميعا

وإن أكرمنا الله ونجحنا ....

لكم منا إن شاء الله شيئا عظيما يستحق قدركم

وإن كانت الاخري فالحمد لله علي كل حال وسنكون من الراضين بقضاء الله .... لأن كل اللي يجي من حبيبي حبيبي

د. محمد السعيد

Spinal muscle atrophy (SMA)

Incidences: It's the second common NM disease after DMD.

Etiology:

It's due to degeneration of AHC of the spinal cord and brain stem.

Types:

→ SMA 1: verdding Hoffman disease.

→ SMA 2: late infantile.

→ SMA 3: juvenile.

C/P:

- Antenatal history: ↓ fetal movement.
- Floppy infant: hypotonia and weakness.
- Paradoxical chest movement . **(مميزة جدا لهذا المرض)**
- Fasciculation mainly in tongue.**(مميزة جدا لهذا المرض)**
- Bulbar symptoms:
  - Hoarseness of voice. **بحة في الصوت**
  - Dysphasia. **صعوبة البلع**
  - dysarthria. **صعوبة الكلام**
  - Nasal regurgitation of fluids. **ارتجاع السوائل من الانف**

N.B.

→ Normal mentality. واعي جدا

→ Normal sensation. حاسس جدا

→ Normal extra ocular muscle. الطفل في العناية لكنه فايق ومركز جدا

Imestigations:

- EMG → muscular fibrillation potentials.
- Muscle biopsy → denervation changes.
- Genetic analysis.
- N.B → CK, NCV are normal.

TTT → plysiotherapy , orthopedic support , psyco and mech. Ventilation.

Myathenia Gravis

Definition: Rapid fatigability of skeletal muscle after repetitive movements, due to presence of (Ab) against A.ch. receptors.(الناقل الكيميائي)

Etiology:

- Autoimmune: (Ab Against Ach. Receptors, associated é thyroid diseases. (most common)
- Congenital AR: (absent receptors.)
- Transient: Transplacental passage of Ab. (من الام المريضة)

C/P:

- **Auto immune:**
  - ❖ Easy fatigability é repetitive movement.
  - ❖ Eye are affected → ptosis, diplopia.
  - ❖ Facial weakness, jaw muscles are affected.
  - ❖ Proximal > distal.
  - ❖ Bulbar symptoms → (hoarseness of voice, Nasal regurgitation, Dysphagia and dysarthria **(المفروض حفظناهم)**
- **Congenital**
  - ❖ From birth (No antibodies).
  - ❖ No A.ch receptors.
  - ❖ Permanent é out improvement.
- **Transient:**
  - ❖ Floppy infant.
  - ❖ Weak suckling (need NGT) feeding.
  - ❖ For days or weeks.

- **Clinical test:**

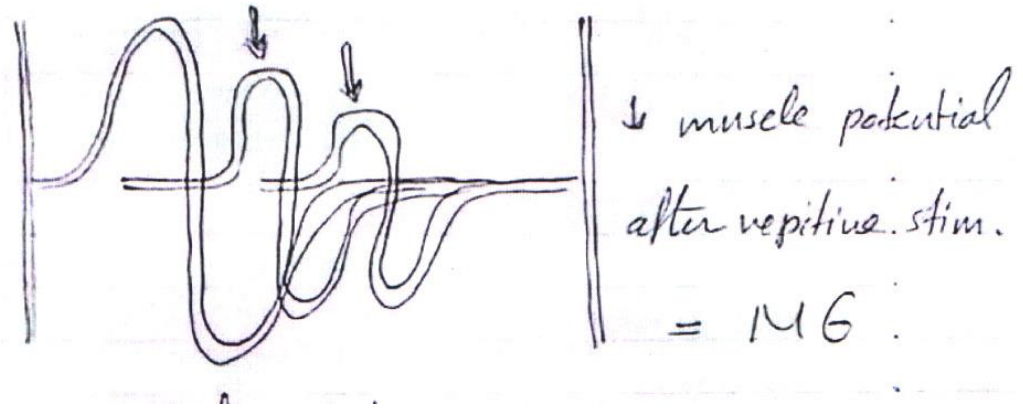
تطلب من العيان ٤ طلبات

ارفع جفنك فوق ....ونزل فكك تحت

ارفع ايدك فوق..... و امسك بقبضة ايدك تحت وللاسف بيفشل في عمل كل ذلك

Investigations:

1- **EMG** → repetitive stimulation →↓ musle potentials.



↓ musele podeutial after repitive stim = MG

2- ↑ anti A.ch reccptors antibodies.

3- ↑ ANA.(لانه مرض مناعي)

4- Thyroid function.(احيانا بيكون مصاب بامراض في الدرقية)

5- Normal CK and NCV

6- Pharmacolog. Lest:

→ Edrophonium → يحدث تحسن لل ptosis. خلال ١٠ ثواني

→ prostigmin → يحدث تحسن لل.ptosis ثواني ٣٠ دقيقة

Treatment:

1- Autoimmune → (steroid, IVIG).

2- Symptomatic → (neostigmins = prostengminve).

3- Ephidrin → ↑ A ch. release.

4- CI → amino glycosides & N.M blockers.**(الجنتاميسين والسكسينيل كولين)**

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Botulism

Organism: Grame +ve anerobic bacteria called (clostridium botulinum)

→ neurotoxin → block of A.ch release .

Mode of transmission:

- Ingestion of canned food. الأطعمة المعلبة
- Wound contamination. تلوث الجروح
- Biological war (terrorism). الارهاب (الحرب البيولوجية)

C/P:

Bulbar symptoms:

- Horseness of voice
- Dysartheria
- Dysphagia
- nasal regurgitation .

Cranial nerve affection

يعني ممكن يحصل فيها ptosis وكمان diplopia تمام زي MG.

Fatigability after repetitive movement

يعني ممكن يحصل فيها تعب في العضلات بعد الحركة المتكررة تمام زي MG.

Constipation (early sign)

Investigations:

- EMG → increase potential after repetitive stimulation.
- Serum → botulinum toxin
- Stool → botulinum toxin or organism.

M.B. → normal CK & NVC

Treatment:

- Botulinium antitoxin.
- Response support (mech. Vent.)
- Nutritional support (NGT)
- A void aminoglycosides .

Peripheral Neuropathy

Definition: (Inflammation of peripheral or cranial nerves.)

Classifications

Etiologically

Hereditary:
- Hereditary. M.S.N.
- Roussy levy synd.

Nutritional:
- Vit B deficiency.
- Pellagrd 3D.

Endocrine:
- DM.
- Thyroid (↑ and ↓)

Metabolic:
- Renal failure.
- Leucodystrophy.

Infective:
- Diphtheria.
- G.B.S.

Neoplasmic: lymphoma.

Clinically

Motor:
- LMNL.
- Distal > proximal.
- Poilaternal .
- LL > UL.
- Foot drop = heigh steppage gait.

Sensory:
- Pain
- Parathesia
- Superficial sensory loss
- Deep sensory loss

Autonomic:
- Vasomotor
- Cutaneous

Pathologically

Degeneration of the axon	Demyelination

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Hereditary Motor Sensory Neuropathy

Peroneal muscle Atrophy

Etiology:

- AD.
- It's the most common genetic neuropathy.

C/P:

1- Motor:

- Weakness, wasting of leg muscles and the lower 1/3 of the thigh.
- Marked discrepancy between Weakness, wasting.
- Skeletal anomalies → pes carus.
- Clumsy gait & falling.
- Foot drop.

2- Sensory:

- Affect fibers of vibration, proprioception.

3- Autonomic:

- Poor vaso motor coutrol → cold feet.

Invvestigations:

- ↓↓ nerve conduction velocity.
- Sural nerve biopsy:
  - ✓ Axonopathy & myelinopathy.
  - ✓ Anion bulb appearance. قلب البصلة

TTT:

- Stabilization of the ankle. with AFO حذاء طبي
- Protection of leg from trauma.

Peroneal muscle atrophy II:

As type 1 but:

Milder than type 1.

Slowly progressive.

Sural biopsy → Axonopathy.

Peroneal muscle atrophy III:

As type 1 but:

More severe than type 1.

Rapidly progressive.

Sural biopsy → more anion bulb.

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Bell's palsy شلل العصب السابع

Definition: It's acute isolated facial nerve paralysis.

Etiology:

- Post infectious immune response.
- Develops abruptly 2 wks after HSV, HZV infection.

C/P

1- Absence of forehead wrinkles.
2- Absence of nasolabial fold.
3- Inability to raise eye brow.
4- Inability to close the eye.
5- Deviation of the angle of the mouth. to healthy side.
6- Drippling of saliva . يريل
7- Inability to (whistle.) يصفر
8- Inability to show the teeth. يكشر

ملحوظة :
يتم فقد الاحساس في اللسان في الثلثين الاماميين علي نفس الناحية المصابة بالشلل.

D.D

- Cong. Facial nerve palsy → absent facial nucleus.
- Neoplastic → neurofibroma, cerebello pontine angle.
- Infarction.
- Traumatic → as birth injury

Treatment

- Prednisolone : 1ng/kg/day for 1 wk → decrease gradually.
- Eye protection: exposure keratitis.
- Acyclovir: (HZV/HSV.)
- Physiotherapy for chronic cases.

Prognosis

- Complete recovery in > 85% of cases
- Mild facial weakness of 10%
- Severe facial weakness of 5%

Guillain-Barre syndrome

Etiology

Post infectious polyneuropathy, usually after non specific viral infection.

Pathology

Dehyelenation → (myelnopathy)

Degeneration of axon → (axonopathy)

C/P

Gradual onset, progressive course.

Weakness, paralysis affecting LL → ascending to trunk → UL → resp. muscles.

Bulbar symptoms → (4)

بحة في الصوت - صعوبة البلع - صعوبة الكلام - ارتجاع السوائل من الانف

Sensory → parasthesia. **اضطراب الاحساس**

Autonomic manifestation → temp, HR, Bl.P

Miller –fisher syndrome (**Areflexia - Ataxia - Ophthalmoplegia**)

Cong. GBS → (hypotonia , Areflexia **,** floppy infant)

Investigations

CSF: It's diagnostic (Increase proteins & decrease cells)

(cytoalbuminous dissociation) وهذه الظاهرة تسمي

NCV→ decrease nerve conduction

EMG → denervation pattern

Serology → campylobacter jejuni, mycoplasma.

Treatment :

Acute phase:

- Hospital admission.(في الطوارئ)
- Supportive measures (resp support , nutritional support).
- IVIG: 400 mg/kg/day. لمدة ٥ ايام
- Plasmapheresis.
- Steroids are **not** effective.

Chronic cases:

- IVIG : 400 mg/Kg/day.
- Plasmapheresis
- Steroids **are** effective.

Prognosis:

- Complete recovery in 85% of patients in 2-3 wks in descending manner.
- Residual weakness in 10%.

Causes of bad prognosis:

- Cranial nerve affection.
- Bulbar symptoms.
- Intubation.
- Maximum disability.

Cause of death: (Respiratory failure)

Familial daysautonomia

Riley – day syndrome

Etiology:

AR sensorimotar neuropalthy → (Automatic dysfunction)

Pathdogy:

- ↓ number for small unmyelinated nerve fibres → loss of pain, temp. taste.
- Number of large myelinated nerve fibres→ loss of deep sensation

C/P:

1- Autonomic:

- Temp. instability & ↑ sweating (الجبهة)
- ↓ tear & Corneal ulcers (العين)
- Instability of blood . Pressure. (القلب)
- ↑ Br. Secretions → pneumonia.(الصدر)

2- Sensory:

- Loss of pain sensation , temperature.
- Deep sensory loss → clumsy gait. يمشي متطوح

3- C.N.S

- Recurrent attacks of seizures.
- MR.

4- Neonatal:

- Floppy infant & Difficult berating.

Investigation:

→ ECG: long QT interval.

→ Chest X-ray: recurrent chest infection

→ EEG: seizures.

→ ID histamine : No Erythema & no pain.

→ ↓ urinary vanilly mardilic acid.

TTT:

- Eye protection from ulcer.
- ttt of chest infection.
- ttt of seizures.
- الثلاثي المرح → Physiotherapy. علاج طبيعي
  → Respiratory support. تنفس صناعي
  → Nutritional support. المساعدة في التغذية

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Duchenne muscle dystrophy

- **Incidence:** The most common NM disease.
- **Etiology:**
  - X-linked recessive disease. (carrier الاولاد مرضي والبنات)
  - Due to mutation of the gene responsible for dystrophin formation that acts as connector between muscle filaments and surrounding muscle cells.
- **C/P**
  - Infancy: asymptomatic or mild hypotonia.
  - Childhood:
    - LMNL: hypotonia, hyporflexia, pseudo hypertrophy.
    - Bil. Symmet. UL, LL Prox > distal weakness.
    - Winging of scapula. بسبب ضعف عضلات الكتف
    - Waddling gait. بسسب ضعف عضلات الحوض
    - Lmbar lordosis بسبب ضعف عضلات الظهر
    - Gower sign. الولد مش قادر يقوم من الارض غير بصعوبة
  - Cordiomyopathy.
  - Increase intellectual function.
- **Investigations:**
  - Increased creatine kinase. وبيكون زيادة كبيرة وبالألاف
  - EMG → myopathic changes.
  - Nerve conduction velocity normal.
  - Increase AST لان هذا الانزيم مش بس في الكبد لأ .... كمان في العضلات
  - Genetic analysis.
  - Muscle biopsy → decrease dystrophin.
- **Treatment:**

Physiotherapy & nutritional support, glucocorticoids.

UMNL & LMNL

Feature	UMNL	LMNL
Site	Cerebral , cerebellum, brain stem, or spinal cord	AHC, roots, nerves, N-M or muscles
Ms. Weakness	Quadriplegia, hemiplegia.	Proximal (myopathy) distal (neuropathy)
Tone	Hypertonia - hyperreflexia	Hypotomia - hyporeflexia
Fasciculation	Absent	Present
Abdom . reflex	Absent	Present
Sensory loss	Cortical sensation : - tactile localization - two points discrimination - stereognosis	Peripheral Sensation as: - pain - touch - pressure - temp

DD of inability to walk

Primary	Secondary
▪ **Paralytic:** • **Brain:** - CP - Hydrocephalus - Bran damage ▪ **Spinal cord:** - Poliomyelitis - Spinal ms. Atraply - Birth trauma - Meuingo myelocele - Spina bifida ▪ **Non paralytic:** - Meutal reterdatio - Bone disease OI - Sever malnutrition - Cong. Myopathies	▪ **Paralytic:** • **Brain**: - Post. Meningitis - Post. Encephalitis - Hydrocephalus. • **Spinal card:** - Transverse myelitis. - Spinal card trauma. - Spinal card trmour. - Neuropathies. - N.M disorders - Myopathies ▪ **Non paralytic** - Clironic infection - Malignancies. - Renal failure

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Acute flaccid paralysis

الشلل الرخـو الحـاد

Definition:

- ❖ It's clinical syndrome characterized by rapid onset of weakness of limbs, may be muscles of respiration and swallowing.
- ❖ The term flaccid means absence of signs of CNS lesion as hypertonia or hyper reflexia.

Causes:

1. Brain stem stroke and encephalitis.
2. Infection of A.H.C
   - Poliomyelitis
   - Non polio enterovirus
3. Acute transverse myelitis.
4. Peripheral neuropathy:
   - Guillian – barre syndrome.
   - Post rabies vaccine neuropathy.
   - Diphtheric neuropathy
   - Heavy metals or drug toxicity.
5. N.M disorders:
   - Myasthenia gravis.
   - Botulism.
   - Tick paralysis.
6. Muscular disorders:
   - Inflammatory myopathy.
   - Viral myositis.
   - Periodic paralysis.

↑ I C P

Def: clinical manifestations of increased ICP occurs when pressure > 25 mmHg . (normal pressure = 0-15 mmHg . or 0-200 mmH2O.)

Etiology :

A) increase cerebral blood flow :(HTN - increased CO2 - decreased O2)

B) hydrocephalus : increased production or decreased absorption .

C) Pathological masses : (Abscess – Hge)

D) Brain edema :

- cytotoxic (CNS infection – HIE)
- Vasogenic (CNS infection – pathological masses)
- Interstitial (hydrocephalus)

C/P :

A) Bulging AF.

B) Papilledema.

C) Neurological (Hypertonia – Hyperreflexia - Hyperventilation)

D) Nonspecific (Headach – vomiting)

E) Cerebral ischemia – convulsions.

Investigations :

- CT Brain.
- Measurement of ICP

TTT :

A) Rapidly acting measures :

(Head elevation 30 * & Hyperventilation & Mannitol 20 %)

B) Slowly acting measures :

(Fluid restriction 60-70 % of maintainance & Furosemide &Steriods)

Brain Tumors

Incidence : The 2nd most common malignancy in children.

Peak age : 5-10 yrs .

Predisposing Factors :

- Neurocutaneous $
- Immunodeficiency $ (Ataxia telangiectasia)

C/P :

A) increased ICP :

- Infancy : Bulging AF – Progressive head enlargement .
- Older children : Headache – vomiting – blurring of vision .

B) False localizing signs :

- Convergent Squint .
- Changes in personality , mentality & speech .

C) True localizing signs :

* **Frontal Lob :** Hemiplegia – focal seizures .
* **Parietal Lob :** Cortical sensory loss – sensory seizures .
* **Temporal Lob :** Auditory agnosia – Focal seizures .
* **Occipital Lob :** contralateral hemianopia – Visual hallucination
* **Cerebellar Ataxia .**
* **Cranial nerve affection .**
* **Pituitary tumors :**

1- Hormonal manifestations :

- Gigantism & acromegaly & Hypopituitarism.

2- Compression manifestations. :

- Optic nerve >> Ipsilateral visual loss.
- Optic chiasma >> Bitemporal hemianopia.

Infratentorial tumors

*** Cerebellar astrocytoma**

*** Medulloblastoma**

*** Brain stem glioma**

*** Ependymoma**

Supratentorial tumors

1- Craniopharyngioma

- **Incidence : most common supratentorial tumor**
- **Consistency** : solid & cystic areas
- Calcification is common
- **C/P :**
  - Focal neurological signs
  - Endocrinal (DI)
  - Increase ICP .
- **TTT** : Surgery + Radiotheraby.

2- Cerebellar Astrocytoma

- **Incidence : most common brain tumor**
- **Consistency** : cystic
- **Site** : unilateral (one cerebellar hemisphere .
- **Metastases** : No metastases.
- **Growth** : Slowly growing.
- **C/P** : Ipsilateral ataxia.
- **TTT** : Surgery + Radiotherapy.

3- Medulloblastoma

- **Incidence** : next most common
- **Consistency** : solid
- **Site** : midline (both hemisphere)
- **Metastases** : Extracranial sites
- **Growth** : Rapidly growing
- **C/P** : Ataxia + increase ICP (more rapid)
- **TTT** : Surgery + Radiotherapy + chemotherapy

4- Optic Glioma

5- Leukemia

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hydrocephalus

def:

dilatation of ventricular system due to increased CSF volume with or without increase in CSF pressure.

CSF circulation:

lateral ventricle (choroid plexus) >>> foramina of monro>>> 3rd ventricle >>> aqueduct of sylvius >>> 4th ventricle >>> foramina of luscka >> subarachnoid

Classification:

1.communicating hydrocephalus

increase formation

(choroid plexus papilloma)

decrease absorption

(sub arachnoid hge – postmeningitic - leukemia - arnold-chiari malformation type 2)

2. obstructive hydrocephalus

congenital

(stenosis of aqueduct - aneurysm of vein of Galen.

Dandy walker malformation .

Acquired

(post meningitis – post. hge – tumor - abcess)

C/P

1. in infancy (befor suture closure)

a) General examination

- ☆ macrocephaly.
- ☆ bulging anterior fontanelle .
- ☆ separated sutures.
- ☆ skull percussion .
- ☆stretched scalp skin .
- ☆sunset sign.

b) Neurological examination

- ☆ delayed developmental milstone .
- ☆ hypertonia .
- ☆ hyperreflexia.
- ☆ weakness .
- ☆ optic nerve atrophy.

2. in older children(after partial sutur closure)

- ☆ head enlargment but less evident.
- ☆ neurological examination as befor.
- ☆ manifestation of increase ICP are prominent.

Investigation:

1) skull x-ray: ☆separation of suture ☆silver beaten appearance

2) cranial u/s, CT, MRI brain.

3) investigations of cause.

TTT

Medical >>> acetazolamide(10-15 mg /kg/day)

Surgical >>> shunt:

- ventriculoperitoneal .
- ventriculo atrial .
- external ventricular drainage.

complication

- infection.
- obstruction.
- shunt nephritis.

D.D >>> causes of macrocephaly

Prognosis

- precosious puperty: increase GNRh.
- visual: vision loss and field defect.
- intellectual: decrease IQ.
- complication of ttt.

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Cerebellar ataxia

Def: Incoordination of volantary motor activity.

C/P:

1- Gait→ a) archicerebellar lesion = drunken gait

b) Neocerebellar lesion

2-Hypotonia & hyporeflexia .

3-Incoordination of voluntary movement:

- Nystagmus.
- staccato speech.
- kinetic tremors.
- rebound phenomena.
- dysmetria.
- dysdiadokokinesia .

Types

Acute ataxia

1- Infection

- Acute Post infectious.Cerebellitis.
- cause : vzv, influnza, enterovirus.
- sudden onset, regressive course .
- Diagnosis: by exclusion & MRI.
- prognosis: excellent.
- age : from 1-3 years preciding varicella infection (2-3w)

2- Toxic.

3- Traumatic.

4- Neoplastic.

5- vascular.

6- migraine.

7- Miller-fisher synd .

B) Chronic Ataxia:

Etiology:

1- congenital anomalies

- cerebellar aplasia.
- Dandy walker $.
- chiari-malformation.
- encephalocele.

2- perinatal : CP

3- Neoplastic

4- Hredofamelial:

- ataxia telangectesia .
- friedrich ataxia.
- Roussy levy $ -degenrative brain dse.

5- Metabplic:

- Refsun disease.
- Abetalipoproteine.
- vit E def.
- Gaucher disease .
- Neimann pick.
- hartnupe .
- maple synp .
- argeno succinic aciduria.

Friedrich ataxia

Etiology:

AR (Triplet repeat expantion)

C/P

- degeneration of Cerebellar ataxia + 3P.

Cerebellum : cerebellar ataxia

Posterior Column : sensory ataxia.

Peripheral nerves : PN.

Pyramidal tract : +ve Babinski & hypotonia.

Generalized seizures

Def:

seizures involving the whole body at the onset of convulsions.

1- Absence seizures

A- typical absence seizures (petit-mal epilepsy)

- age:5-8 yrs
- form: sudden loss of consciousness & cessation of activity.
- frequency:5-30 /da.y
- duration: few seconds.
- precipitated by hyperventilation.
- EEG:generalized spike.

B- atypical absence seizures:

- absence + myoclonic seizures

2- Generalized tonic-clonic seizures (grand mal):

The most common & ccc by prescence of 3 stages:

a-aura: motor,sensory,autonomic or psychic

b- seizures:

- sudden loss of consciousness.
- tonic phase:falling to the ground,apnea,cyanosis.
- clonic phase:rhythmic ms contraction & relaxation,loss of sphincter &.
- post-ictal: headache, lethargy, Todd's paralysis

3- Infantile spasm :

Etiology:

1- Idiopathic.

2- Symptomatic:80%.

- Prenatal:congenital anomalies,infection,radiation

- Natal:HIE,birth injury.

- Postnatal:CNS infection,trauma,ICH,↑↑ Na, ↓↓Glu.

, ↓↓Na, kernicterus

Form

Onset: infancy-

Type:symmetrical contraction of neck,trunk,extremities-

- occur ,shortely after getting up from sleep

- 3types: flexor, extensors or mixed spasms

EEG: hypsarrhythmia

TTT:ACTH,steroids,vigabatrin.

4- Landau - Kleffner syndrome

Age: 5yrs + seizures disorder + loss of language skills &aphasia.

Partial seizures (focal)

Def:

seizures starting in one part of the body (± loss of consciousness)

1-simple: without impairment of consciousness

a -motor

- aura (epigastric discomfort,visual,sensory)

- tonic or clonic

Consciousnss: intact-

Automatism:**absent**-

Duration:10-20 sec-

b - Sensory: pain,parasthesia

c-Autonomic:nausea,vomiting, palpitation .

d-Psychic: smell, taste or emotion.

2-complex (motor only) : focal with impairment of consciousness.

- aura (epigastric discomfort, visual, sensory),

- tonic or clonic.

Consciousness: intact-

- automatic repetitive movements present

- Duration:few minutes

- MRI: temporal lesions.

3-partial seizures with 2ry generalization:

It is progress from partial to generalized seizures.

4-benign epilepsy with centrotemporal spikes (Rolandic)

- age:6-12yrs
- form:parasthesia,tonic or clonic,icrease salivation,dysphagia
- seizures occur during sleep(75%)

Consciousnss: usually intac -

- EEG:centrotemporal spike
- Prognosis:excellent.

5- Rasmussen encephalitis:

- Etiology:autoantibodies stimulate glutamate receptors
- C/P:unilateral intractable partial seizures
- EEG:diffuse brain insult with slow background activity
- Progosis:poor

Treatment of epilepsy

General prinsiples

1- anticonvulsant drug is not indicated after one seizure with normal ECG.

2- the drug of choice depends on classification of seizure.

3 - start with one drug then increase the dose before adding another drug.

4- blood screening of the drug is done every month .

5- duration of anticonvulsant therapy is 2 years - free seizures.

6- weanining is done over 3-6 months.

Common anti convulsants :

1- phenobarbitone (G. tonic – clonic & partial & status epilepticus)

2- phenytoin. (G. tonic – clonic & partial & status epilepticus)

3- Carbamazepine (G. tonic – clonic & partial)

4- Na valproate : broad spectrum .

5- Clonazepam adjuvant ttt

6- Ethosuximide (absence srizures .)

Status Epilepticus

Def:

continuous convulsions more than 30 min or serial convulsions without regain of consciousness inbetween.

Incidence:

10% in children with epilepsy.

30% as 1st presentation of epilepsy.

Etiology:

1- prolonged febrile convulsions (common.)

2- idiopathic

3- symptomatic :

a - CNS lesions: encephalitis , meningitis, hypoxia, cong. Malformations.

b - metabolic : IEM (PKU), electrolyte disturbances

TTT:

1-first aid measures: Airway, 02, IV or rectal diazepam.

2- Full lap (ABG , electrolytes ,CBC ,liver ,kidney function)

3- CT scan, continous EEG.

4-LP, toxicological ,IEM screening, MRI

5-ICU admission, vital signs, ryle, IV line.

6- anticonvulsants:

a- IV diazepam (0.05 - 0.1mg/kg)

b- Phenytoin: 15 - 20mg / kg loading, 5mg/kg/day maintainance

c- If phenytoin not effective → phenobarbital can be used instead.

d-Intubation may be needed.

In refractory status (not responding to 1st line ttt)

-IV paraldehyde.

-thipental + MV.

-IV sodium vaproate.

-continous midazolam infusion.

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